Brain and Spine 4 (2024) 102718

Contents lists available at ScienceDirect

Brain and Spine

journal homepage: www.journals.elsevier.com/brain-and-spine

Management of patients with a neurovascular conflict involving the optic nerve and a non-diseased intracranial artery: Three cases

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ARTICLE INFO

Handling editor: W Peul

Keywords:
Neurovascular conflict
Opticopathy
Microsurgical decompression
Optic nerve
Anterior cerebral artery
Carotid artery

ABSTRACT

Introduction: Determining whether a neurovascular conflict (NVC) involving the anterior visual pathway (AVP) and a non-diseased intracranial artery is amenable for microvascular decompression is challenging. Moreover, it is unclear whether microvascular decompression of the optic nerve is an effective therapy.
Research question: What are the outcomes of different treatment strategies for NVCs involving the AVP and a non-diseased intracranial artery?
Material and methods: Data on patients with symptomatic NVCs involving the AVP and a non-diseased intracranial artery was collected and included treatment and outcome parameters. The case series was drafted in accordance with the CARE guidelines.
Results: Three patients aged 53,53 and 55 visited our out-patient clinic with a suspected symptomatic NVC between the optic nerve and a non-diseased intracranial artery. A conservative treatment was opted for in the first patient aimed at treating her glaucoma, with temporary improvement of symptoms. Microvascular decompression of the optic nerve was performed in two patients. One operated patient developed post-operative complications resulting in posterior circulation perfusion decline, while the other experienced a worse tunnel vision with a decrease in visual acuity.
Discussion and conclusion: The diagnosis of a symptomatic NVC between the AVP and a non-diseased intracranial artery should be considered with caution, i.e. after exclusion of all other causes. Microvascular decompression can be performed but does not necessarily improve symptoms. A better understanding of the pathophysiological mechanisms underlying these NVCs is warranted to determine the benefit of microvascular decompression of the optic nerve.

1. Introduction

Visual symptoms such as visual field deficits and lowered visual acuity are occasionally caused by intracranial compression of the anterior visual pathway (AVP). In most instances, the compression is caused by a neoplasm or vascular malformation (aneurysms, dolichoectatic arteries) (Madill and Riordan-Eva, 2004). A less frequently recognized cause of compression of the AVP can be a neurovascular conflict (NVC) involving a non-diseased intracranial artery such as the internal carotid artery (ICA) or anterior cerebral artery (ACA). Such NVC can entail a mere contact of an artery with the AVP without distortion of the optic nerve (ON) contours or compression of the AVP with displacement, impression or grooving of the ON. Either form can cause a neurovascular compression syndrome (Jacobson et al., 1997), (Gelkopf et al., 2023).

The prevalence of NVCs between the AVP and non-pathological arteries is unknown, however, recent radiological literature suggests that this phenomenon could be relatively common, ranging from 11% to 70% (compression with distortion of the ON versus contact with the AVP without distortion), and is usually asymptomatic (Jacobson et al., 1997; Tsutsumi et al., 2017). It is therefore difficult, and perhaps controversial, to attribute visual symptoms to a radiologically identified NVC involving the AVP. Additionally, this is further complicated by the concomittant presence of ophthalmological conditions presenting with similar symptoms.

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https://doi.org/10.1016/j.bas.2023.102718
Received 5 July 2023; Received in revised form 16 November 2023; Accepted 25 November 2023
Available online 29 November 2023
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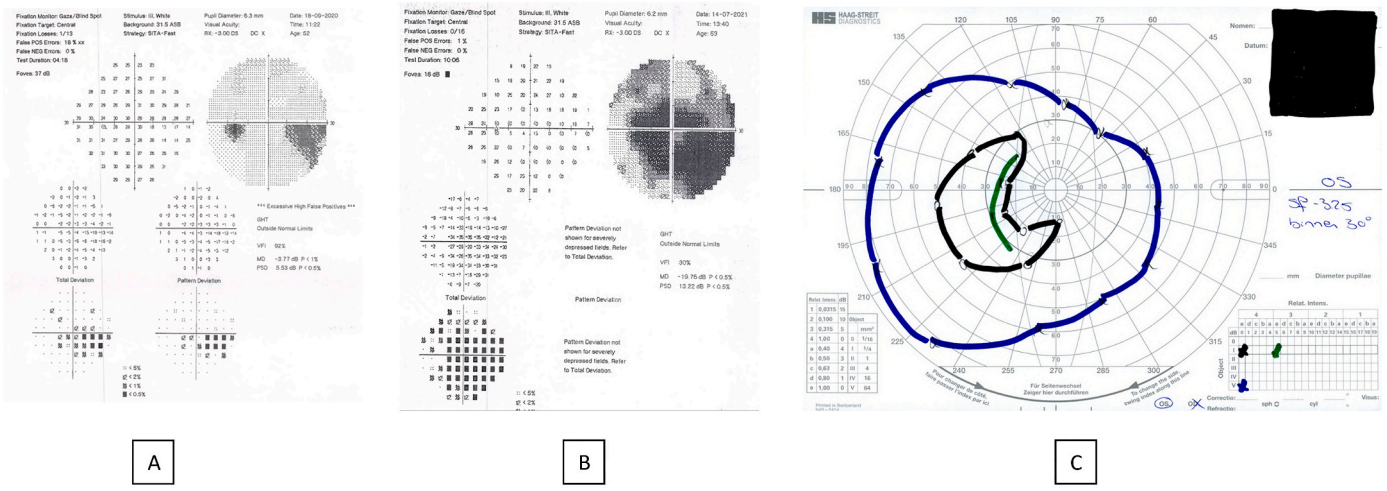

Fig. 1. Visual field examinations of patient 1
Abbreviation: OS: oculus sinister.
A: Humphrey field analyzer results OS performed in 2020 showing a lower nasal visual field defect. B: Humphrey field analyzer results OS performed in 2021 showing progression of the visual field defect with a significant central scotoma. C: Goldman OS performed in 2023 showing temporal sparing of the visual field: isopter V4–I4–I3.

Consequently, estimating whether a symptomatic patient with a NVC involving the AVP may benefit from surgical decompression remains challenging. To shed further light on these challenges and surgical outcome, we present three cases of patients with visual symptoms and the presence of contact between the AVP and a healthy ICA or ACA with different management strategies and outcomes. Additionally, we have reviewed literature on the diagnosis, management and outcome of rare symptomatic NVC involving the AVP and a non-diseased ICA. Ultimately, we aim to provide an overview of how this rare NVC is diagnosed and how treatment strategies are justified.

2. Methods

This case series was drafted in accordance with the CARE guidelines for adequate reporting of case series (Gagnier et al., 2013). In accordance with local ethical regulations, informed consent was obtained from all patients.

3. Case presentations

3.1. Case 1

3.1.1. Clinical presentation and work-up

A 53-year-old female with a medical history of asthma and myopia regularly visited the ophthalmological outpatient clinic for management of a bilateral open-angle glaucoma. In 2019, intraocular pressure (IOP) was 18–27 mmHg (normal range: 10–21 mmHg) in the right eye, and 21–29 mmHg in the left eye. In 2020, the patient's visual acuity (VA) on the left eye decreased from 1.0 to 0.5 with IOPs of 18–45 mmHg, while VA of the right eye remained stable, with unchanged IOPs. A relation with the patient's use of hydrocortisone creme and steroid inhaler was suspected and steroid therapy was therefore discontinued. Pharmacological therapy was further tailored to improve IOP regulation, with initially satisfying results. In 2021, VA of the left eye deteriorated to 0.15 with a recurrent increase in IOP on the left side rising to 36 mmHg. Further physical examination revealed a cup-disc ratio of 0.9, a left-

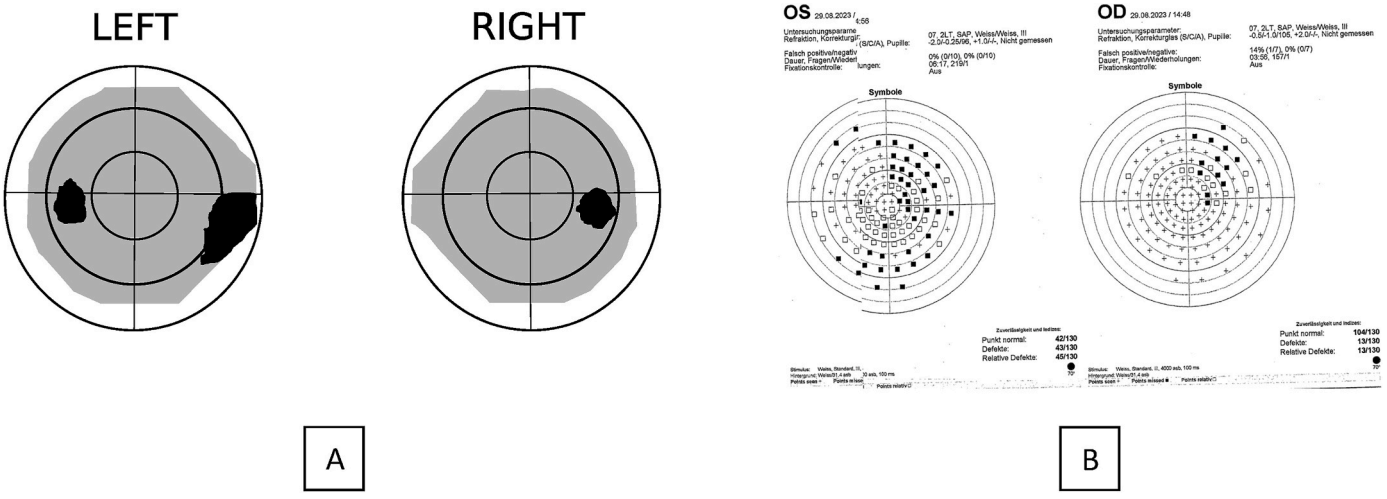

Fig. 2. Visual field examinations of patient 2
Abbreviation: OS: oculus sinister.
A: Schematic representation of the pre-operative visual fields, showing a nasal deficit in OS. B. Visual field examination 41 months after surgery showing a right homonymous superior quadrantanopia.

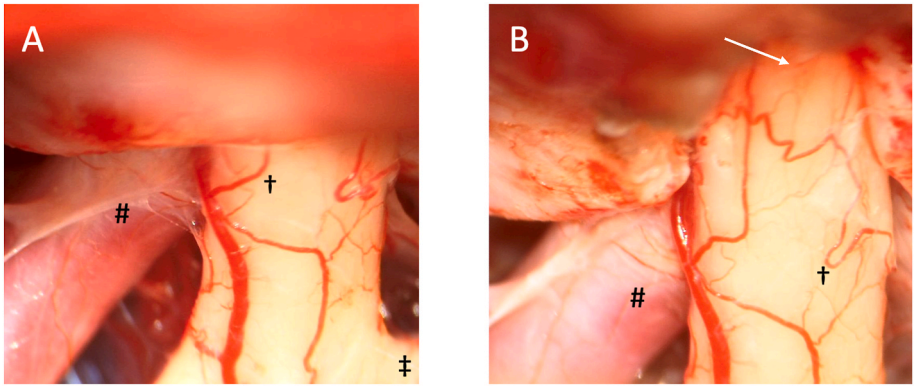

Fig. 3. Intraoperative image of patient 2 and regions of interest.
Abbreviations: ICA: internal carotid artery, OC: optic chiasm, ON: optic nerve.
Pre- (A) and post- (B) decompression with the left ICA (#), left ON (†) and the OC (‡) with clear unroofing of the bony the optic canal (white arrow).

sided relative afferent pupillary defect (RAPD), and an impaired color vision on the left. Visual field examination revealed a central scotoma and nasal deficit on the left side (Fig. 1). An additional MRI of the brain was performed because of pronounced unilateral symptomatology, relatively young age, and fast progression of symptoms. This MRI revealed contact between the ACA and the left side of the optic chiasm, without clear compression of the AVP (Fig. 6A).

3.1.2. Management and outcome

Both patient and neurosurgeons opted for a conservative treatment as the causal relationship between the NVC and the visual symptoms could not be ascertained. Moreover, most symptoms were attributed to a progressive glaucoma pathology. From 2021 onwards, visual field deficits and VA remained relatively stable, until the patient developed severe allergic reactions to the eye drops. Eye drops were discontinued and IOP in the left eye increased to 50 mmHg. A PAUL glaucoma implant was placed, resulting in a temporary decrease in IOP and a stable visual field.

3.2. Case 2

3.2.1. Clinical presentation and work-up

A 53-year-old, otherwise healthy, woman presented with a six-month history of visual disturbances that were diagnosed as a nasal visual field deficit to the left eye. Visual field examination using Humphrey field analyzer (HFA) revealed a nasal scotoma (Fig. 2). IOP was low (15-15 mmHg), and VA as well as color vision were undisturbed. The unusual scotoma without morphological correlations suggested a compressive optic neuropathy and an MRI of the brain was performed. The MRI revealed a thinned left ON in close vicinity to the ipsilateral ophthalmic segment of a normally configured non-sclerotic ICA (Fig. 6B).

3.2.2. Management and outcome

In the absence of conservative treatment options and the patient's work as an illustrator that was troubled by the visual field deficit, surgical microvascular decompression was offered. A left pterional craniotomy was performed and a sub-frontal approach was used to access the optic cistern. An upward pulsation of the left sided ICA towards the ipsilateral ON was observed and caused intermittent compression of the ON (Fig. 3A). Decompression was achieved by unroofing the bony optic canal and splitting the falciform ligament, allowing the ON to divert from the ICA pulsations (Fig. 3B). On postoperative day one, the patient mentioned subjective visual improvement. Several hours later, the patient went into cardiac arrest. After successful resuscitation, she was diagnosed with a severe pulmonary embolism. Due to prolonged hemodynamic instability, systemic intravenous thrombolysis was necessary in addition to intravenous heparinization. Several hours later, the intubated patient presented with a Glasgow Coma Scale of 8 and anisocoria. Subsequent CT showed transtentorial herniation due to ipsilateral acute subdural hematoma that required emergent surgical evacuation. Visual field deficits of the left eye worsened and were accompanied by additional deficits to the right eye, most likely as a result of temporary herniation compromising the posterior circulation (Fig. 4). At forty-one months after surgery, the patient reported subjective improvements compared to after the emerency decompression but visual field examination showed a persistent right homonymous superior quadrantanopia (Fig. 2).

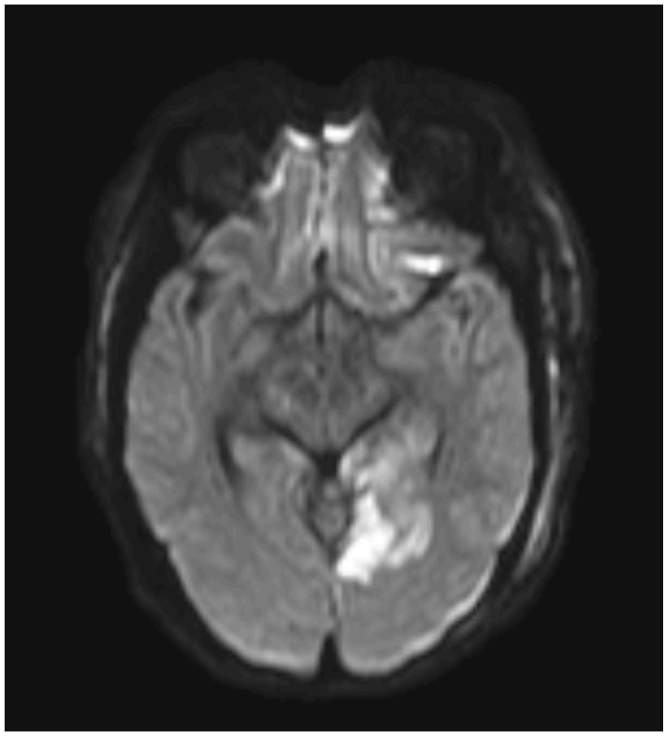

Fig. 4. MRI-DWI of the brain of patient 2 with restricted diffusion in the left occipital lobe suggestive of ischemia.
Abbreviation: MRI-DWI: magnetic resonance imaging – diffusion-weighted imaging.

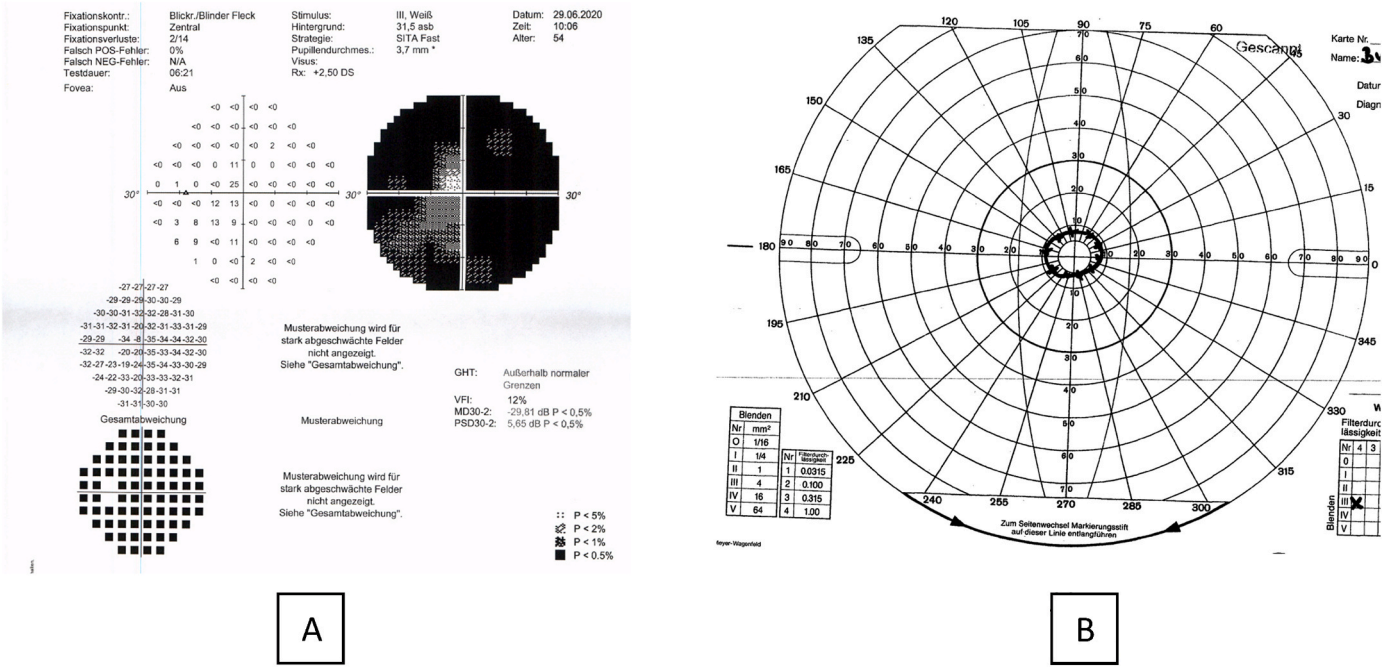

Fig. 5. Visual field examinations of patient 3
Abbreviation: OD: oculus dexter.
A: Humphrey field analyzer results OD performed in 2020 showing a significant tunnel vision. B. Goldmann OD showing persistent significant tunnel vision at 6 months after surgery.

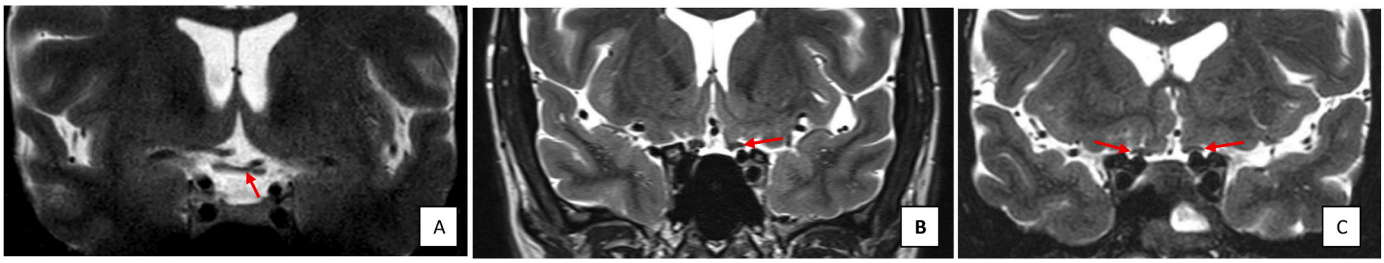

Fig. 6. MRI images of the three cases of patients with neurovascular conflicts involving the AVP and ICA or ACA.
AbbreviationsACA: anterior cerebral artery; AVP: anterior visual pathway; ICA: internal carotid artery; MRI: magnetic resonance imaging; OC: optic chiasms; ON: optic nerve.
A: Left ACA is in contact with the left side of the OC (red arrow) in patient 1. B: Images of patient 2 showing a thinned left ON (red arrow) in close vicinity to the left ophthalmic segment of the ICA. C: Bilateral neurovascular conflict between the ICA and the ON (red arrows) with more pronounced thinning of the ON the left side in patient 3. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

3.3. Case 3

3.3.1. Clinical presentation and work-up

A 55-year-old male, with a history of left-sided amblyopia since birth presented with a progressive loss of VA to the right eye with a sub-acute onset. Visual acuity was 0.63/<0.1 at initial ophthalmological examination. Visual field examination revealed a pronounced tunnel vision (Fig. 5). Relative afferent pupillary reaction and color vision were undisturbed. Optical coherence tomography and funduscopic evaluation were normal and an MRI revealed a bilateral NVC between the ICA and ON with ON thinning that was more pronounced on the left side (Fig. 6C).

3.3.2. Management and outcome

The progression of symptoms in the only functional eye in absence of other causative factors led to interdisciplinary discussion of optic nerve decompression. Microsurgical decompression on the right side was offered and the patient was subsequently treated in technical analogy to the patient described in case 2. Similar to case 2, intermittent compression of the ON by the ICA was observed. Postoperatively, VA slightly deteriorated on the right eye (0.25/0.1), while tunnel vision improved slightly yet persisted. At 6 months, VA improved to 0.4/0.05, but visual field deficits worsened to 5° (Fig. 5). Retrospectively, symptom presentation and dynamics were unusual. After several thorough ophthalmological work-ups including visual evoked potentials (VEPs), a diagnosis of non-organic visual loss was made. With this in mind, the patient may consciously or unconsciously have confounded the examinations.

4. Discussion

We presented three cases of patients with sub-acute progressive impairment of VA and/or visual field defects (Table 1). In all three cases, the atypical clinical course prompted extensive ophthalmological and radiological examination. Moreover, the MRI revealed contact between the AVP and a healthy ICA or ACA. Different management strategies were applied based on case-specific features, illustrating the complexity of decision-making in this disorder.

Table 1
Overview of the clinical characteristics, treatment and outcomes of the three patients with a neurovascular conflict involving the optic nerve and the internal carotid artery or anterior cerebral artery.

	Patient 1	Patient 2	Patient 3
Sex (female/male)	Female	Female	Male
Age (years)	53	53	55
Medical history	Asthma, myopia, open angle glaucoma	None	Amblyopia
Onset visual symptoms	Sub-acute (few months)	Sub-acute (few months)	Sub-acute (few weeks)
Progressive (yes/no)	Yes	Yes	Yes
Length of visual symptoms	18 months	5 months	2 months
First presenting symptom	Loss of visual acuity OS	Blind spot OS	Loss of visual acuity OD
Visual field defect (yes/no)	Yes	Yes	Yes
Location visual field defect	Central/nasal scotoma	Nasal scotoma left 40–50° max.	Concentric
Intraocular pressure (mmHg, R/L)	18–45	15–15	21–18
Visual acuity (R/L)	0.15/1.2	1.0/1.0	0.63/<0.1
Relative afferent pupillary defect (yes/no)	Yes	Unknown	No
Impaired color vision (yes/no)	Yes	No	No
Reason for MRI	Atypical glaucoma presentation, young age	Unclear scotoma without morphological correlations in the ophthalmological examination	Otherwise, unexplained progressive visual impairment
Finding on the MRI	Contact between the ACA and left OC	ICA contact with left ON	Bilateral contact between ICA and ON left > right
Surgery (yes/no)	No	Yes	Yes
Type of surgery	NA	Optic nerve decompression (left)	Optic nerve decompression (right)
Immediate post-operative outcome	NA	Visual field deterioration L > R	Visual acuity 0.25/0.1, better but persistent tunnel vision
Post-operative outcome 6 months	NA	NA	Visual acuity 0.4/0.1, tunnel vision worse again, till 5°
Post-operative outcome 12 months	NA	Subjective improvement but persistent right homonymous superior quadrantanopia	Diagnosed with nonorganic visual loss
Complications	NA	Pulmonary embolism, anticoagulation therapy, acute subdural hematoma with brain herniation needing evacuation	NA

Abbreviations: ACA, anterior cerebral artery; ICA, internal carotid artery; NA, not applicable; OD: oculus dexter; ON, optic nerve; OS: oculus sinister.

4.1. Pathophysiology

The pathophysiology of progressive visual deterioration due to a NVC on the ON is unknown. A well-known example of vascular compression of a cranial nerve is trigeminal neuralgia, usually caused by microvascular compression. Electron microscopy studies of trigeminal rhizotomy specimens from patients with intractable trigeminal neuralgia showed chronic demyelination beneath the region of indentation, and therefore, similar demyelination processes are likely to play a role in NVCs involving the ON (Love et al., 1998). However, as opposed to the optic nerve, symptomatic compression of the trigeminal nerve or the facial nerve usually occurs at the root entry/exit zone. This so-called Redlich-Obersteiner's zone is a transition zone between the central and peripheral nervous system, and histologically characterized by the fact that myelin covering the neurons is produced by Schwann cells (peripheral nervous system) while this is produced by oligodendrocytes within the central nervous system (Szmyd et al., 2022). This transition zone is suspected to be a susceptible zone for demyelination caused by compression. As the optic nerve is part of the central nervous system, it is solely myelinated by oligodendrocytes and lacks a vulnerable transition zone (Mayoral et al., 2018). This anatomical difference may explain the difference in symptomatology and clinical course, i.e. slowly decreasing neuronal signaling versus paroxysmal hyperexcitability, between a compression neuropathy of the optic nerve and trigeminal nerve, respectively. However, clear evidence for this suggestion is still lacking.

4.2. Diagnosis

Diagnosis of a NVC between the AVP and a healthy intracranial artery is established by an MRI. However, proving whether such a NVC causes visual symptoms is an arduous task, requiring an extensive diagnostic work-up. This is related to the fact that 1) contact between the AVP and a healthy artery is often asymptomatic (Jacobson et al., 1997), 2) these types of NVCs are known to cause unspecific symptoms (Jacobson, 1999), and 3) there are often concomitant ophthalmological disorders with overlapping visual symptomatology (Ogata et al., 2005). Regarding the latter, case 1 illustrates this difficulty as the co-existence of high-tension glaucoma hampered the diagnosis of a NVC as cause of the presenting symptoms.

Overlapping symptoms between NVCs involving the AVP and a non-pathological ACA or ICA and ophthalmological disorders, in particular glaucoma, have been described previously and include visual field defects, loss of VA and pallor of the neuroretinal rim (Jacobson, 1999). Moreover, longstanding compression of the ON can cause a nerve fiber bundle pattern of visual field deficits and excavation of the optic disc mimicking glaucomatous opticopathy (Jacobson, 1999). The frequent concomitant presence of NVCs involving the ON and glaucoma suggests a potential etiological relationship. Indeed, Ogata et al. revealed that patients with compression of the ON by the ICA were significantly more likely to have normal tension glaucoma compared to patients without a compression of the ON (Ogata et al., 2005). To the best of our knowledge, we present the first case of a patient with high-tension glaucoma in whom a NVC involving the AVP and the ACA is identified. Despite the presence of symptoms denoting a compressive etiology such as an afferent pupillary reaction defect or impaired color vision, the high IOP also manifested itself as a plausible treatable cause of her visual field defect.

4.3. Natural course and conservative management

Once a NVC involving the AVP and a non-diseased intracranial artery is diagnosed with an MRI of the brain, management consists of either a conservative treatment with regular follow-up or microsurgical decompression. Determining which cases are amenable for surgery is however challenging. One must consider the natural history and progression of the condition and weigh these against the risks and benefits

Table 2
Case-reports describing microvascular decompressions of optic nerves in contact with healthy intracranial arteries.

Author	Year	Symptoms	Physical Exam	LOS	MRI finding	Surgery	Outcome
Nishioka	1995	Progressive visual loss OD	Binasal hemianopia, atrophy right optic disc, swelling left optic disc, VA 20/200 & 20/500	3 months	Downward loop of ACA on OC	Lifting ACA complex	Immediate subjective improvement, VA after 1 month 20/40 both sides, minimal VF improvement
McLaughlin	2011	Bilateral visual loss	Binasal hemianopia, IOP 19torr ODS, bilateral nasal step	8 years	Vascular loop ACA compressing OC with inferior displacement	Teflon between ACA and OC	Improvement VF 48h after surgery, completely resolved VA at 4 months
Strom	2011	Progressive visual loss OS, headache	Temporal defect, OS VA 20/100, color vision Rand Rittler 3/6	2 years	ICA in contact with inferolateral aspect ON, chiasm and tract	Two felts between ON and ICA	VA OS 20/40, color 4/6, complete recovery at 6 months
Andrews	2016	Progressive visual loss OD	Monocular nasal field deficit	Acute	Tortuous supraclinoid ICA displacing ON	Incision arachnoid adhesions, Teflon between ICA and ON	Subjective improvement VF, objective improvement at 6 weeks

Abbreviations: ACA, anterior cerebral artery; ICA, internal carotid artery; IOP: intraocular pressure; LOS, length of symptoms; OC, optic chiasm; OD, oculus dexter; ON, optic nerve; OS: oculus sinister; VA, visual acuity; VF, visual field.

of invasive surgery. The literature on the natural course of AVP compressions by a healthy ACA or ICA is sparse and shows contradicting findings. Unlike tumors or aneurysms that may grow rapidly, NVCs involving the ON and a non-pathological artery is usually considered an insidious process causing slowly progressing symptoms. However, a case series by Jain et al. including 37 patients with radiologically confirmed compression of the AVP by an intracranial artery and visual dysfunction revealed that 24 out of the 26 followed-up patients (96%) had stable VA on follow-up with a mean follow-up of four years (Jain et al., 2019). None of these patients were operated on. The authors concluded that opticopathy from a vascular compression can be recognized by its non-progressive nature. Conversely, in a case-report by Andrew et al., a 36-year-old female patient presented with acute painless monocular visual field deficit, most probably caused by a tortuous course of the supraclinoid segment of the ICA compressing the right ON. The acute presentation of a chronic process could mirror a loss of compensatory mechanism whereby venous insufficiency ultimately leads to micro-infarction (McDougall, 2016). In our cases, all three patients presented with sub-acute (within weeks to months) onset of symptoms that showed progression within months. Thus, the onset and progression of AVP compression by intracranial arteries is highly variable and therefore, careful monitoring of symptoms with regular re-evaluation of treatment options is advised.

4.4. Neurosurgical management

If operative treatment is considered, it usually consists of a pterional or lateral supraorbital craniotomy to explore the relationship between the ON and neighboring vasculature (Strom et al., 2012; McLaughlin and Bojanowski, 2011; McDougall, 2016). Intradural maneuvers may vary depending on intraoperative findings from freeing the nerve in transition to the optic canal, to dissection of arachnoid adhesions to the ON, and insertion of a piece of Teflon® between the ON and the compressing artery. We found four published cases of visual impairments resulting from a NVC involving a non-diseased ICA or ACA and the AVP that were operated on (Table 2). In these four patients, duration of symptoms varied from a few months to many years. Postoperatively, visual symptoms improved in all cases regardless of length of symptoms (Table 2).

It seems plausible, considering the relatively high reported incidence of NVCs involving the AVP and a non-pathological intracranial artery, that neurosurgeons and ophthalmologists are frequently confronted with NVCs involving the AVP and a healthy intracranial artery. The very few, yet positive reports on the outcome of microsurgical decompressions of the ON make this treatment modality appealing. However, publication bias needs to be considered and there may be a lack of reports on negative results of surgical decompression. To date, the literature is too sparse to advocate for surgical decompression. Yet, in cases with continuous deterioration or rapid deterioration of visual symptoms, and following exclusion of other causes, decompressive surgery may be considered.

5. Lessons learned

A few lessons can be learned from the three cases. Regarding case 1, the decision to treat the patient conservatively was based on the concurrent glaucoma, which explained most of her symptoms. The role of the NVC in this disease and symptom presentation remains however unsolved. In this case, the patient's firm decision to refrain from undergoing surgery further supported the decision to treat her conservatively. Regarding surgery, the greatest challenge is determining the indication and timing of surgical decompression. Patient 2 and 3 suffered progressive symptoms over a period of two to five months, which could not be explained by other causes, expediting surgical treatment. However, one could, also considering the usually slowly progressive history of neurovascular compression syndromes as reported in the literature, favor a wait-and-scan policy to get a more accurate and objective impression of the progression of symptoms. Besides this, it could have been informative to perform VEPs before surgery, as this may raise questions regarding the NVC as cause of the visual symptoms. Ultimately, the management strategy for this pathology entails careful weighing of the benefits, risks and patient-specific considerations. More importantly, patients should be well counselled about the potential lack of improvement following decompression and risks associated with intracranial surgery.

6. Limitations

Limitations of this case series include its retrospective nature and small population size (n = 3). Patient 2 suffered from post-operative complications influencing her vision and therefore, the effect of surgery is difficult to evaluate. Lastly, our discussion is mainly based on other small case series and case reports with a potential publication bias as mentioned above.

7. Conclusion

Neurovascular conflicts involving the AVP and non-pathological intracranial arteries are common radiological findings and occasionally present with visual symptoms. Diagnosis is per exclusionem and therefore requires extensive ophthalmological, neurological and radiological work-up. The natural course varies, further impeding clinical decision making. We emphasize the need to report on management outcomes, both conservative and neurosurgical, to stimulate our understanding of the disease and to improve clinical management and decision-making.

Conflict of interest

There are no conflicts of interest to declare.

Declaration of Competing interest

There are no conflicts of interest to declare.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bas.2023.102718.

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